Transfer of antioxidant message by microvesicles mediates antiapoptotic effects on human endothelial cells

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Type de publication	Communication
Type	Communication avec actes dans un congrès
Année	2013
Date du colloque	22-24/04/2013
Titre du colloque	17ème Congrès de Physiologie, Pharmacologie et Thérapeutique
Titre des actes ou de la revue	Fundamental & Clinical Pharmacology : Abstracts of the 17th Annual Meeting of French Society of Pharmacology and Therapeutics
Volume	27, Suppl 1.
Pagination	79
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Pays	France
Editeur	Wiley
Ville	Angers
Mots-clés	Apoptosis [5], Endothelium [6], microvesicles [7], Oxidative Stress [8]
Résumé en anglais	Microvesicles (MVs) are small membrane-derived fragments shed from various cell types during activation and/or apoptosis and represent a new class of biological information mediators. MVs generated from T lymphocytes undergoing activation and apoptosis exert a beneficial potential effect on the cardiovascular system through their dual capacity to increase nitric oxide and reduce reactive oxygen species production. This study investigated the effect of MVs on the apoptosis of human umbilical vein endothelial cells triggered by actinomycin D (Act D). The effect of Act D treatment on reactive oxygen species (ROS) production was biphasic. Indeed, ROS levels significantly increased during the early phase of apoptosis (2 h) and after 10 h of treatment, but not at 4, 8 and 24 h. MVs significantly attenuated the increase in ROS production induced by Act D at 2 h, but not at 10 h, indicating that they normalize ROS production during the early phase of apoptosis by acting directly as ROS scavengers, owing to their ability to carry active antioxidant enzymes such as catalase and the three isoforms of the superoxide dismutase. Furthermore, the effects of MVs on the late phases of apoptosis were associated with the ability of these vesicles to increase the expression of manganese-superoxide dismutase, probably by the transfer of its mRNA, in endothelial cells, through internalization process. These findings illustrate new mechanisms by which MVs from T lymphocytes exert their vasculo-protective effects by improving endothelial function under pathological conditions in which apoptosis and oxidative stress are enhanced.

URL de la notice	http://okina.univ-angers.fr/publications/ua12140 [9]
DOI	10.1111/fcp.12026 [10]

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